

FILE 'HCAPLUS' ENTERED AT 14:21:28 ON 12 DEC 2008

L1 5354 S BETA GLUCAN
L2 575595 S ANTIBODY OR ANTIBODIES OR IMMUNOGLOBULIN OR IGG
L3 228947 S YEAST OR ZYMOSAN
L4 185259 S BRANCHED OR BRANCHING OR BRANCH
L5 354 S L1 AND L2
L6 102 S L1 AND L2 AND L3
L7 5 S L1 AND L2 AND L3 AND L4
L8 877426 S CANCER OR TUMOR OR NEOPLA?
L9 102 S L1 AND L2 AND L3 AND L6
L10 27 S L1 AND L2 AND L3 AND L8
L11 12 S L10 AND (PY<2005 OR AY<2005 OR PRY<2005)

=> file hcaplus		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

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FILE COVERS 1907 - 12 Dec 2008 VOL 149 ISS 25
 FILE LAST UPDATED: 11 Dec 2008 (20081211/ED)

HCAplus now includes complete International Patent Classification (IPC) reclassification data for the third quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s beta glucan
      1575694 BETA
      16591 GLUCAN
L1      5354 BETA GLUCAN
          (BETA(W)GLUCAN)

=> s antibody or antibodies or immunoglobulin or IGG
      339895 ANTIBODY
      409341 ANTIBODIES
      32650 IMMUNOGLOBULIN
      81605 IGG
L2      575595 ANTIBODY OR ANTIBODIES OR IMMUNOGLOBULIN OR IGG

=> s yeast or zymosan
      223053 YEAST
      6172 ZYMOSAN
L3      228947 YEAST OR ZYMOSAN

=> s branched or branching or branch
      85085 BRANCHED
      60380 BRANCHING
      51986 BRANCH
L4      185259 BRANCHED OR BRANCHING OR BRANCH

=> s l1 and l2
L5      354 L1 AND L2

=> s l1 and l2 and l3
L6      102 L1 AND L2 AND L3
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=> s 11 and 12 and 13 and 14
L7 5 L1 AND L2 AND L3 AND L4

=> d 17 1-5 ti abs bib

L7 ANSWER 1 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Oral administration of a new soluble branched
 β -1,3-D-glucan is well tolerated and can lead to increased salivary
concentrations of immunoglobulin A in healthy volunteers
AB The soluble branched yeast β -1,3-D-glucan (SBG)
belongs to a group of carbohydrate polymers known to exert potent
immunomodulatory effects when administered to animals and humans. A new
oral solution of SBG has been developed for local application to the
oropharyngeal and esophageal mucosa in order to strengthen the defense
mechanisms against microbial and toxic influences. In the present study
oral administration of SBG has been investigated primarily for assessment
of safety and tolerability in an early phase human pharmacol. study (phase
I). Eighteen healthy volunteers were included among non-smoking
individuals. The study was an open 1 : 1 : 1 dose-escalation safety study
consisting of a screening visit, an administration period of 4 days and a
follow-up period. Groups of six individuals received SBG 100 mg/day, 200
mg/day or 400 mg/day, resp., for 4 consecutive days. The dose increase
was allowed after a careful review of the safety data of the lower dose
group. No drug-related adverse event, including abnormalities in vital
signs, was observed. By inspection of the oral cavity only minor mucosal
lesions not related to the study medication were seen in seven subjects.
Repeated measurements of β -glucan in serum
revealed no systemic absorption of the agent following the oral doses of
SBG. In saliva, the IgA concentration increased significantly for the highest
SBG dose employed. SBG was thus safe and well tolerated by healthy
volunteers, when given orally once daily for 4 consecutive days at doses
up to 400 mg.
AN 2006:111575 HCAPLUS <<LOGINID::20081212>>
DN 145:20695
TI Oral administration of a new soluble branched
 β -1,3-D-glucan is well tolerated and can lead to increased salivary
concentrations of immunoglobulin A in healthy volunteers
AU Lehne, G.; Haneberg, B.; Gaustad, P.; Johansen, P. W.; Preus, H.;
Abrahamsen, T. G.
CS Clinical Research Unit, Rikshospitalet-Radiumhospitalet Trust, Oslo,
Norway
SO Clinical and Experimental Immunology (2006), 143(1), 65-69
CODEN: CEXIAL; ISSN: 0009-9104
PB Blackwell Publishing Ltd.
DT Journal
LA English
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Solubilized cell wall β -glucan, CSBG, is an
epitope of Candida immune mice
AB Antibody to β -glucan is generally
difficult to produce in mice. The authors have recently developed a
protocol to obtain a soluble Candida spp. β -(1 \rightarrow 3)-D-Glucan
(CSBG) by sodium hypochlorite (NaClO) oxidation and subsequent DMSO (Me₂SO)
extraction. CSBG is composed mainly of β -(1 \rightarrow 3) and
 β -(1 \rightarrow 6)-glucosidic linkages with a small amount of
branch. In this paper, mice were immunized with Candida albicans
and the specificity of the resulting sera to CSBG was examined by ELISA.

Using CSBG coated plate, sera of the Candida immune mice showed higher reactivity than non-immune, normal mice and the reactivity was neutralized by adding soluble CSBG as a competitor. However, the reactivity could not be neutralized by a β -(1 \rightarrow 6) branched β -(1 \rightarrow 3)-glucan, grifolan. Similar specificity of the sera was obtained by com. available β -glucan particle, zymosan or zymocel, immune mice. These facts strongly suggested that CSBG included epitopes of the specific antibody in Candida immune mice.

AN 2000:311223 HCAPLUS <<LOGINID::20081212>>

DN 133:72623

TI Solubilized cell wall β -glucan, CSBG, is an epitope of Candida immune mice

AU Uchiyama, Michiharu; Ohno, Naohito; Miura, Noriko N.; Adachi, Yoshiyuki; Tamura, Hiroshi; Tanaka, Shigenori; Yadomae, Toshiro

CS Laboratory for Immunopharmacology of Microbial Products, School of Pharmacy, Tokyo University of Pharmacy and Life Science, Tokyo, 192-0392, Japan

SO Biological & Pharmaceutical Bulletin (2000), 23(5), 672-676

CODEN: BPBLEO; ISSN: 0918-6158

PB Pharmaceutical Society of Japan

DT Journal

LA English

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN

TI PGG-Glucan, a soluble β -(1,3)-glucan, enhances the oxidative burst response, microbicidal activity, and activates an NF- κ B-like factor in human PMN: Evidence for a glycosphingolipid β -(1,3)-glucan receptor

AB PGG-Glucan, a soluble β -(1,6)- branched β -(1,3)-linked glucose homopolymer derived from the cell wall of the yeast *Saccharomyces cerevisiae*, is an immunomodulator which enhances leukocyte anti-infective activity and enhances myeloid and megakaryocyte progenitor proliferation. Incubation of human whole blood with PGG-Glucan significantly enhanced the oxidative burst response of subsequently isolated blood leukocytes to both soluble and particulate activators in a dose-dependent manner, and increased leukocyte microbicidal activity. No evidence for inflammatory cytokine production was obtained under these conditions. Electrophoretic mobility shift assays demonstrated that PGG-Glucan induced the activation of an NF- κ B-like nuclear transcription factor in purified human neutrophils. The binding of 3H-PGG-Glucan to human leukocyte membranes was specific,

concentration-dependent, saturable, and high affinity (Kd.apprx.6 nM). A monoclonal antibody specific to the glycosphingolipid lactosylceramide was able to inhibit activation of the NF- κ B-like factor by PGG-Glucan, and ligand binding data, including polysaccharide specificity, suggested that the PGG-Glucan binding moiety was lactosylceramide. These results indicate that PGG-Glucan enhances neutrophil anti-microbial functions and that interaction between this β -glucan and human neutrophils is mediated by the glycosphingolipid lactosylceramide present at the cell surface.

AN 1999:112996 HCAPLUS <<LOGINID::20081212>>

DN 130:351132

TI PGG-Glucan, a soluble β -(1,3)-glucan, enhances the oxidative burst response, microbicidal activity, and activates an NF- κ B-like factor in human PMN: Evidence for a glycosphingolipid β -(1,3)-glucan receptor

AU Wakshull, Eric; Brunke-Reese, Deborah; Lindermuth, Johanna; Fisette,

Leslie; Nathans, Robin S.; Crowley, John J.; Tufts, Jeffrey C.; Zimmerman, Janet; Mackin, William; Adams, David S.

CS Department of Biology, Alpha-Beta Technology, Worcester, MA, 01605, USA

SO Immunopharmacology (1999), 41(2), 89-107

CODEN: IMMUDP; ISSN: 0162-3109

PB Elsevier Science B.V.

DT Journal

LA English

RE.CNT 89 THERE ARE 89 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Interrelation of structure and antitumor effects of fungal (1→3)
β-D-glucans.

AB In the last 25 yr chemical and pharmacol. studies have been focused on the non-cytotoxic, immunomodulating polysaccharides. Yeast and related fungal (1→3)-β-D-glucans, especially, those having appropriate O-6-β-D-glucosyl branches (db, 1/3 to 1/5) exhibited strong antitumor effects, and can be used as an immunostimulator in cancer therapy. Such antitumor effects may be due to the triple helix of the backbone; (1→6)-β-glucan of lichen and also synthetic branched (1→4)-β-D-glucans were inactive. In addition, our extensive studies on the structure-activity relationship using various branched (1→3)-β-D-glucans (db, 1/25 - 3/4) showed that the distribution of the branches along the backbone and their mol. shapes may also play a role in expression of antitumor activity, as indicated by modification of the side chains. We will discuss interrelation of structure and antitumor effects of immunomodifying glucans, e.g., an exocellular glucan of Pestalotia sp (db, 3/5), and a highly active glucan (db. 1/4) from Volvariella volvaceas, and also antibody specificities of Volvariella glucan.

AN 1996:412276 HCAPLUS <<LOGINID::20081212>>

TI Interrelation of structure and antitumor effects of fungal (1→3)
β-D-glucans.

AU Misaki, A.; Kakuta, M.; Kishida, Etsu

CS Faculty Human Life Science, Osaka City University, Sumiyoshi, 558, Japan

SO Book of Abstracts, 212th ACS National Meeting, Orlando, FL, August 25-29 (1996), CARB-042 Publisher: American Chemical Society, Washington, D. C. CODEN: 63BFAF

DT Conference; Meeting Abstract

LA English

L7 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Effect of structurally different yeast β-glucans on immune responses in Atlantic salmon (Salmo salar L.)

AB The immunostimulatory effects of different yeast β-glucans in Atlantic salmon were studied in three sets of expts. First, the different β-glucans were assessed for their ability to induce an increase in blood lysozyme activity after i.p. injection. Second, the same glucans were included in an exptl. furunculosis vaccine, where their adjuvant effects on antibody response against the bacterial antigen were examined. Finally, the ability of the glucans to prime the respiratory burst response of salmon macrophages was investigated. In an earlier study it was demonstrated that of two different yeast β-glucans, Macro-Gard (previously known as M-Glucan) was significantly more potent in protecting Atlantic salmon against bacterial pathogens than the other called DL-Glucan. The present study showed that the principal structural differences between these two yeast β-glucans were the presence of β-1,6-linked chains in MacroGard which were absent in DL-Glucan, and the more frequent branching

in MacroGard compared to DL-Glucan. With respect to immunostimulatory effects, MacroGard was more effective in inducing responses than DL-Glucan in all three sets of expts. By studying the effects of MacroGard particles treated chemical or enzymically to remove β -1,6-linkages, the authors found that the β -1,6-linked chains did not seem to be important for the immunostimulatory effect. It was demonstrated that the majority of side chains in MacroGard were β -1,3-linked and, furthermore, the results indicated that the number of β -1,3-linked side chains may be decisive for the immunostimulatory effect of yeast β -glucan in Atlantic salmon.

AN 1996:125403 HCAPLUS <<LOGINID::20081212>>
 DN 124:198499
 OREF 124:36631a,36634a
 TI Effect of structurally different yeast β -glucans on immune responses in Atlantic salmon (*Salmo salar* L.)
 AU Engstad, Rolf E.; Robertsen, Boerre
 CS Norwegian College Fishery Science, University Tromso, Tromso, N-9037, Norway
 SO Journal of Marine Biotechnology (1995), 3(1-3, Proceedings of the Third International Marine Biotechnology Conference, 1994), 203-7
 CODEN: JMBOEW; ISSN: 0941-2905
 PB Springer
 DT Journal
 LA English

=> s cancer or tumor or neopla?
 385201 CANCER
 477055 TUMOR
 571101 NEOPLA?
 L8 877426 CANCER OR TUMOR OR NEOPLA?

=> s l1 and l2 and l3 and l6
 L9 102 L1 AND L2 AND L3 AND L6

=> d his

(FILE 'HOME' ENTERED AT 14:20:55 ON 12 DEC 2008)

FILE 'HCAPLUS' ENTERED AT 14:21:28 ON 12 DEC 2008

L1 5354 S BETA GLUCAN
 L2 575595 S ANTIBODY OR ANTIBODIES OR IMMUNOGLOBULIN OR IGG
 L3 228947 S YEAST OR ZYMOSAN
 L4 185259 S BRANCHED OR BRANCHING OR BRANCH
 L5 354 S L1 AND L2
 L6 102 S L1 AND L2 AND L3
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 L8 877426 S CANCER OR TUMOR OR NEOPLA?
 L9 102 S L1 AND L2 AND L3 AND L6

=> log hold

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FULL ESTIMATED COST	25.31	25.52
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
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CA SUBSCRIBER PRICE	-4.00	-4.00

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FILE 'HCAPLUS' ENTERED AT 14:30:12 ON 12 DEC 2008
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5117814 AY<2005
4593999 PRY<2005
L11 12 L10 AND (PY<2005 OR AY<2005 OR PRY<2005)

=> d l11 1-12 ti abs bib

L11 ANSWER 1 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Drug delivery product and methods
AB The present invention provides a particulate delivery system comprising an extracted yeast cell wall comprising β -glucan, a payload mol., and a payload trapping mol. The invention further provides methods of making and methods of using the particulate delivery system.
AN 2005:1335040 HCAPLUS <<LOGINID::20081212>>
DN 144:74766
TI Drug delivery product and methods
IN Ostroff, Gary R.
PA USA
SO U.S. Pat. Appl. Publ., 45 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	US 20050281781	A1	20051222	US 2004-869693	20040616 <--
	CA 2570313	A1	20060119	CA 2005-2570313	20050615 <--
	WO 2006007372	A2	20060119	WO 2005-US21161	20050615 <--
	WO 2006007372	A3	20060921		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ,				

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 NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,
 SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU,
 ZA, ZM, ZW
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 EP 1755567 A2 20070228 EP 2005-786318 20050615 <--
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 IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA,
 HR, LV, MK, YU
 CN 101001615 A 20070718 CN 2005-80019946 20050615 <--
 BR 2005012068 A 20080206 BR 2005-12068 20050615 <--
 JP 2008503472 T 20080207 JP 2007-516687 20050615 <--
 US 20060083718 A1 20060420 US 2005-230017 20050919 <--
 MX 2006PA14552 A 20070523 MX 2006-PA14552 20061213 <--
 IN 2007KN00163 A 20070629 IN 2007-KN163 20070112 <--
 PRAI US 2004-869693 A 20040616 <--
 US 2004-610872P P 20040917 <--
 WO 2005-US21161 W 20050615

L11 ANSWER 2 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Frequency-assisted transdermal agent delivery method and system

AB The invention discloses an apparatus and method for transdermally delivering a
 biol. active agent comprising a delivery system having a microprojection
 member (or system) that includes a plurality of microprojections (or array
 thereof) that are adapted to pierce through the stratum corneum into the
 underlying epidermis layer, or epidermis and dermis layers, a formulation
 containing the biol. active agent and an oscillation-inducing device. In one
 embodiment, the biol. active agent is contained in a biocompatible coating
 that is applied to the microprojection member. In a further embodiment,
 the delivery system includes a gel pack having an agent-containing hydrogel
 formulation that is disposed on the microprojection member after
 application to the skin of a patient. In an alternative embodiment, the
 biol. active agent is contained in both the coating and the hydrogel
 formulation.

AN 2005:614580 HCAPLUS <<LOGINID::20081212>>

DN 143:139175

TI Frequency-assisted transdermal agent delivery method and system

IN Chan, Keith T.; Cormier, Michel J. N.; Lin, WeiQi

PA USA

SO U.S. Pat. Appl. Publ., 24 pp.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 20050153873	A1	20050714	US 2004-971441	20041021 <--
	AU 2004314416	A1	20050804	AU 2004-314416	20041021 <--
	WO 2005069758	A2	20050804	WO 2004-US34923	20041021 <--
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 SN, TD, TG

BR 2004017757 A 20070410 BR 2004-17757 20041021 <--
 JP 2007519446 T 20070719 JP 2006-549239 20041021 <--
 PRAI US 2004-535275P P 20040109 <--
 WO 2004-US34923 W 20041021 <--

L11 ANSWER 3 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Cancer therapy using β -glucan and
 monoclonal antibodies

AB The invention provides methods for using neutral soluble glucan and
 monoclonal antibodies for antitumor therapy. Neutral soluble
 β (1,3; 1,6) glucan enhances the tumoricidal activity of the innate
 immune system by binding to the C3 complement protein receptor CR3. The
 glucan does not stimulate the induction of inflammatory cytokines. Also
 described are methods of using whole glucan particles as an
 immunomodulator by inducing a shift from a Th2 response to the Th1
 response, leading to an enhanced antitumor cytotoxic T-cell response.

AN 2004:308355 HCAPLUS <<LOGINID::20081212>>

DN 140:297492

TI Cancer therapy using β -glucan and
 monoclonal antibodies

IN Ross, Gordon D.

PA University of Louisville Research Foundation, Inc., USA

SO PCT Int. Appl., 92 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004030613	A2	20040415	WO 2003-US27975	20030904 <--
	WO 2004030613	A3	20050113		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
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	CA 2496508	A1	20040415	CA 2003-2496508	20030904 <--
	AU 2003295326	A1	20040423	AU 2003-295326	20030904 <--
	EP 1539194	A2	20050615	EP 2003-786508	20030904 <--
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	CN 1694715	A	20051109	CN 2003-824893	20030904 <--
	CN 1939335	A	20070404	CN 2006-10136269	20030904 <--
	US 20060009419	A1	20060112	US 2005-526185	20050803 <--
PRAI	US 2002-408126P	P	20020904	<--	
	CN 2003-824893	A3	20030904	<--	
	WO 2003-US27975	W	20030904	<--	

L11 ANSWER 4 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Cancer therapy using whole glucan particles and
 antibodies

AB The present invention relates to methods of using whole glucan particles
 and complement activating antibodies for antitumor therapy.

Whole glucan particles enhance the tumoricidal activity of the innate immune system by binding to the C3 complement protein receptor CR3. This binding enhances innate immune system cytotoxicity, as well as stimulating the release of activating cytokines.

AN 2004:220160 HCAPLUS <<LOGINID::20081212>>

DN 140:247055

TI Cancer therapy using whole glucan particles and antibodies

IN Ostroff, Gary R.; Ross, Gordon D.

PA Biopolymer Engineering, Inc., USA; University of Louisville Research Foundation, Inc.

SO PCT Int. Appl., 62 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004021994	A2	20040318	WO 2003-US27841	20030904 <--
	WO 2004021994	A3	20040812		
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	AU 2003268486	A1	20040329	AU 2003-268486	20030904 <--
	EP 1536820	A2	20050608	EP 2003-749452	20030904 <--
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK			
	CN 1694715	A	20051109	CN 2003-824893	20030904 <--
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	CN 2003-824893	A3	20030904	<--	
	WO 2003-US27841	W	20030904	<--	

L11 ANSWER 5 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Collaborative induction of inflammatory responses by dectin-1 and Toll-like receptor 2

AB Toll-like receptors (TLRs) mediate recognition of a wide range of microbial products including lipopolysaccharides, lipoproteins, flagellin, and bacterial DNA, and signaling through TLRs leads to the production of inflammatory mediators. In addition to TLRs, many other surface receptors have been proposed to participate in innate immunity and microbial recognition, and signaling through some of these receptors is likely to cooperate with TLR signaling in defining inflammatory responses. In this report we have examined how dectin-1, a lectin family receptor for β -glucans, collaborates with TLRs in recognizing microbes. Dectin-1, which is expressed at low levels on macrophages and high levels on dendritic cells, contains an immunoreceptor tyrosine-based activation motif-like signaling motif that is tyrosine phosphorylated upon activation. The receptor is recruited to phagosomes containing zymosan particles but not to phagosomes containing IgG-opsonized particles. Dectin-1 expression enhances TLR-mediated

activation of nuclear factor κ B by β -glucan-containing particles, and in macrophages and dendritic cells dectin-1 and TLRs are synergistic in mediating production of cytokines such as interleukin 12 and tumor necrosis factor α . Addnl., dectin-1 triggers production of reactive oxygen species, an inflammatory response that is primed by TLR activation. The data demonstrate that collaborative recognition of distinct microbial components by different classes of innate immune receptors is crucial in orchestrating inflammatory responses.

AN 2003:368316 HCAPLUS <<LOGINID::20081212>>

DN 138:384005

TI Collaborative induction of inflammatory responses by dectin-1 and Toll-like receptor 2

AU Gantner, Benjamin N.; Simmons, Randi M.; Canavera, Scott J.; Akira, Shizuo; Underhill, David M.

CS Department of Immunology, University of Washington, Seattle, WA, 98105, USA

SO Journal of Experimental Medicine (2003), 197(9), 1107-1117

CODEN: JEMEAV; ISSN: 0022-1007

PB Rockefeller University Press

DT Journal

LA English

RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 6 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells

AB A method of treatment of disease by inhibition of cellular secretory processes is provided. The method has particular application in the treatment of diseases dependent on the exocytotic activity of endocrine cells, exocrine cells, inflammatory cells, cells of the immune system, cells of the cardiovascular system, and bone cells. Agents and compns. therefor, as well as methods for manufacturing these agents and compns., are provided. In a preferred embodiment a clostridial neurotoxin, substantially devoid of holotoxin binding affinity for neuronal cells of the presynaptic muscular junction, is associated with a targeting moiety. The targeting moiety is selected such that the clostridial toxin conjugate so formed may be directed to a non-neuronal target cell to which the conjugate may bind. Following binding, a neurotoxin component of the conjugate, which is capable of inhibition of cellular secretion, passes into the cytosol of the target cell by cellular internalization mechanisms. Thereafter, inhibition of secretion from the target cell is effected.

AN 2001:228744 HCAPLUS <<LOGINID::20081212>>

DN 134:247267

TI Clostridial neurotoxin targeted conjugates for inhibition of secretion from non-neuronal cells

IN Foster, Keith Alan; Chaddock, John Andrew; Purkiss, John Robert; Quinn, Conrad Padraig

PA Microbiological Research Authority, UK

SO PCT Int. Appl., 63 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	WO 2001021213	A2	20010329	WO 2000-GB3669	20000925 <--
	WO 2001021213	A3	20020711		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,			

HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
 LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
 SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
 ZA, ZW
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
 DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
 CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
 CA 2383470 A1 20010329 CA 2000-2383470 20000925 <--
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 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO, MK, CY, AL
 JP 2003509476 T 20030311 JP 2001-524636 20000925 <--
 AU 782457 B2 20050728 AU 2000-74365 20000925 <--
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 AU 2005227383 A1 20051124 AU 2005-227383 20051027 <--
 AU 2005227383 B2 20080821
 PRAI GB 1999-22554 A 19990923 <--
 WO 2000-GB3669 W 20000925 <--

L11 ANSWER 7 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN
 TI Immunopharmacological and immunotoxicological activities of a
 water-soluble (1 → 3)-β-D-glucan, CSBG from Candida spp
 AB We have established a convenient, two-step procedure to solubilize the
 yeast cell wall (1→3)-β-D-glucan using the combination
 of NaClO oxidation and DMSO extraction Candida soluble β-D-glucan (CSBG) was
 mainly composed of a linear β-1,3 glucan with a linear
 β-1,6-glucan moiety. In this study, we screened for several
 immunopharmacol. activities of CSBG and found the following activities:
 (1) interleukin-6 synthesis of macrophages in vitro; (2) antagonistic
 effect for zymosan mediated-tumor necrosis factor
 synthesis of macrophages; (3) augmentation for lipopolysaccharide mediated
 tumor necrosis factor and nitrogen oxide syntheses of macrophages;
 (4) activation of alternative pathway of complement; (5) hematopoietic
 response on cyclophosphamide induced leukopenia; (6) the antitumor effect
 on ascites form tumor; (7) Enhanced vascular permeability; (8)
 priming effect on lipopolysaccharide triggered TNF-α synthesis; and
 (9) adjuvant effect on antibody production These results strongly
 suggested that CSBG possessed various immunopharmacol. activity.
 AN 2000:235041 HCAPLUS <<LOGINID::20081212>>
 DN 133:12504
 TI Immunopharmacological and immunotoxicological activities of a
 water-soluble (1 → 3)-β-D-glucan, CSBG from Candida spp
 AU Tokunaka, Kazuhiro; Ohno, Naohito; Adachi, Yoshiyuki; Tanaka, Shigenori;
 Tamura, Hiroshi; Yadomae, Toshiro
 CS Laboratory for Immunopharmacology of Microbial Products, School of
 Pharmacy, Tokyo University of Pharmacy and Life Science, Tokyo, 192-0392,
 Japan
 SO International Journal of Immunopharmacology (2000), 22(5),
 383-394
 CODEN: IJIMDS; ISSN: 0192-0561
 PB Elsevier Science Ltd.
 DT Journal
 LA English
 RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN
 TI Interactions of Penicillium marneffei with human leukocytes in vitro
 AB Penicillium marneffei, a dimorphic fungus endemic in parts of Asia, causes
 disease in those with impaired cell-mediated immunity, especially persons with
 AIDS. The histopathol. of penicilliosis marneffei features the

intracellular infection of macrophages. The authors studied the interactions between human leukocytes and heat-killed yeast -phase *P. marneffei*. Monocyte-derived macrophages bound and internalized *P. marneffei* in the presence of complement-sufficient pooled human serum (PHS). Binding and phagocytosis were still seen if PHS was heat inactivated or omitted altogether. The binding of unopsonized *P. marneffei* to monocyte-derived macrophages occurred in the absence of divalent cations and was not affected by inhibitors of mannose and . beta.-glucan receptors or monoclonal antibodies directed against CD14 and CD11/CD18. Binding was profoundly inhibited by wheat germ agglutinin. A vigorous respiratory burst was seen in peripheral blood mononuclear cells (PBMC) stimulated with *P. marneffei*, regardless of whether the fungi were opsonized. However, tumor necrosis factor alpha (TNF- α) release from PBMC stimulated with *P. marneffei* occurred only if serum was present. These data demonstrate that (i) monocyte-derived macrophages bind and phagocytose *P. marneffei* even in the absence of opsonization, (ii) binding is divalent cation independent but is inhibited by wheat germ agglutinin, suggesting that the major receptor(s) recognizing *P. marneffei* is a glycoprotein with exposed N-acetyl- β -D-glucosaminyl groups, (iii) *P. marneffei* stimulates the respiratory burst regardless of whether opsonins are present, and (iv) serum factors are required for *P. marneffei* to stimulate TNF- α release. The ability of unopsonized *P. marneffei* to parasitize mononuclear phagocytes without stimulating the production of TNF- α may be critical for the virulence of this intracellular parasite.

AN 1999:554591 HCAPLUS <<LOGINID::20081212>>

DN 131:285214

TI Interactions of *Penicillium marneffei* with human leukocytes in vitro

AU Rongrungruang, Yong; Levitz, Stuart M.

CS The Evans Memorial Department of Clinical Research and the Department of Medicine, Boston University School of Medicine, Boston, MA, 02118, USA

SO Infection and Immunity (1999), 67(9), 4732-4736

CODEN: INFIBR; ISSN: 0019-9567

PB American Society for Microbiology

DT Journal

LA English

RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 9 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Antigen-specific response of murine immune system toward a yeast β -glucan preparation, zymosan

AB Zymosan, a particulate β -glucan preparation from *Saccharomyces cerevisiae*, shows various biol. activities, including anti-tumor activity. We have previously shown that soluble . beta.-glucan initiated anti-tumor activity was long-lived and was effective even by prophylactic treatment at 1 mo prior to tumor challenge. However, the activity by zymosan was relatively short-lived. Antigen-specific responses of mice to zymosan might be a causative mechanism. In this paper, mice were immunized with zymosan and antibody production and antigen-specific responses of lymphocytes to zymosan were analyzed. Sera of zymosan immune mice contained zymosan -specific IgG assessed by ELISA and FACS. Spleen and bone marrow cells of zymosan-immune mice showed higher cytokine production in response to zymosan. Specificity of zymosan -specific responses were also analyzed using various derivs. prepared from zymosan. These facts strongly suggested that mice recognize zymosan as antigen in addition to non-specific immune stimulant.

AN 1999:311543 HCAPLUS <<LOGINID::20081212>>

DN 131:128740

TI Antigen-specific response of murine immune system toward a yeast
 β -glucan preparation, zymosan
AU Miura, T.; Ohno, N.; Miura, N. N.; Adachi, Y.; Shimada, S.; Yadomae, T.
CS School of Pharmacy, Laboratory for Immunopharmacology of Microbial
Products, Tokyo University of Pharmacy and Life Science, Hachioji, Tokyo,
192-0392, Japan
SO FEMS Immunology and Medical Microbiology (1999), 24(2), 131-139
CODEN: FIMIEV; ISSN: 0928-8244
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 10 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Targeting of natural killer cells to mammary carcinoma via naturally
occurring tumor cell-bound iC3b and β -
glucan-primed CR3 (CD11b/CD18)
AB Previous reports have suggested that malignant cells frequently generate a
humoral immune response that is ineffective in tumor
destruction. Despite coating tumors with IgM and IgG that
activate the C system via the classical pathway, normal membrane
regulators of C (e.g., membrane cofactor protein and CD59) prevent
cytotoxicity. Moreover, C3 deposition on tumors does not result in
cytotoxic recognition by phagocytes or NK cells bearing C3 receptors
capable of mediating destruction of C3-opsonized bacteria or yeast
. The current investigation showed that freshly excised mammary tumors
bore IgM, IgG, and C3 detectable by flow cytometry. Normal sera
contained natural IgM and IgG Abs reactive with breast
tumor cell lines, and IgG Ab titers were increased in
patients with breast cancer. Breast tumor cell lines
incubated in normal serum from AB+ individuals activated the classical,
but not the alternative, pathway of C and became coated with C3. Despite
exhibiting membrane-bound C3, serum-opsonized breast tumor cell
lines were not killed by CR3 (CD11b/CD18)-bearing NK cells. Priming of NK
cell CR3 with small soluble yeast β -glucan
polysaccharides enabled CR3-dependent killing of these same C3-bearing
tumor cell lines. Tests of mammary carcinoma cells from freshly
excised tumors demonstrated that they also bore sufficient amts. of
opsonic C3 for cytotoxic recognition by NK cells bearing
polysaccharide-primed CR3, whereas they were largely resistant to NK cells
bearing unprimed CR3. This study demonstrates the potential utility of
using naturally occurring opsonic C3 on tumor cells for specific
immunotherapeutic targeting by NK cells and phagocytes bearing
polysaccharide-primed CR3.
AN 1997:448273 HCAPLUS <<LOGINID::20081212>>
DN 127:204305
OREF 127:39698h,39699a
TI Targeting of natural killer cells to mammary carcinoma via naturally
occurring tumor cell-bound iC3b and β -
glucan-primed CR3 (CD11b/CD18)
AU Vetvicka, Vaclav; Thornton, Brian P.; Wieman, T. Jeffery; Ross, Gordon D.
CS Division of Experimental Immunology and Immunopathology, Dep. of Pathology
and Division of Surgical Oncology, Dep. of Surgery, University of
Louisville, Louisville, KY, 40292, USA
SO Journal of Immunology (1997), 159(2), 599-605
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
RE.CNT 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 11 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Interrelation of structure and antitumor effects of fungal (1→3) β -D-glucans.

AB In the last 25 yr chemical and pharmacol. studies have been focused on the non-cytotoxic, immunomodulating polysaccharides. Yeast and related fungal (1→3)- β -D-glucans, especially, those having appropriate O-6- β -D-glucosyl branches (db, 1/3 to 1/5) exhibited strong antitumor effects, and can be used as an immunostimulator in cancer therapy. Such antitumor effects may be due to the triple helix of the backbone; (1→6)- β -glucan of lichen and also synthetic branched (1→4)- β -D-glucans were inactive. In addition, our extensive studies on the structure-activity relationship using various branched (1→3)- β -D-glucans (db, 1/25 - 3/4) showed that the distribution of the branches along the backbone and their mol. shapes may also play a role in expression of antitumor activity, as indicated by modification of the side chains. We will discuss interrelation of structure and antitumor effects of immunomodifying glucans, e.g, an exocellular glucan of *Pestalotia* sp (db, 3/5), and a highly active glucan (db. 1/4) from *Volvariella volvacea*, and also antibody specificities of *Volvariella* glucan.

AN 1996:412276 HCAPLUS <<LOGINID::20081212>>

TI Interrelation of structure and antitumor effects of fungal (1→3) β -D-glucans.

AU Misaki, A.; Kakuta, M.; Kishida, Etsu

CS Faculty Human Life Science, Osaka City University, Sumiyoshi, 558, Japan

SO Book of Abstracts, 212th ACS National Meeting, Orlando, FL, August 25-29 (1996), CARB-042 Publisher: American Chemical Society, Washington, D. C.

CODEN: 63BFAF

DT Conference; Meeting Abstract

LA English

L11 ANSWER 12 OF 12 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Ingestion of acapsular *Cryptococcus neoformans* occurs via mannose and β -glucan receptors, resulting in cytokine production and increased phagocytosis of the encapsulated form

AB *Cryptococcus neoformans* is a pathogenic yeast and a major cause of opportunistic infection in AIDS patients. It is commonly found in an acapsular form in the environment, and infection is likely to occur by inhalation. The lung provides a suitable environment for capsule synthesis, and once encapsulated, *C. neoformans* becomes resistant to phagocytosis. A stable acapsular mutant of the organism is readily ingested by murine macrophages in vitro, indicating entry via constitutively competent receptors. We demonstrate in this report that this process is inhibitable by particles derived from *Saccharomyces cerevisiae* that are rich in mannan and β -glucan, as well as more purified forms of these glycans. Furthermore, ingestion of the acapsular form of *C. neoformans* induces a range of proinflammatory cytokines, including tumor necrosis factor alpha and granulocyte-macrophage colony-stimulating factor, which, as we have previously shown, enhance ingestion of serum-opsonized encapsulated *C. neoformans* in vitro. We demonstrate that ingestion of the acapsular form of the organism also enhances ingestion of the pathogenic encapsulated form. This is dependent on the production of tumor necrosis factor alpha and granulocyte-macrophage colony-stimulating factor by the macrophages, since addition of neutralizing antibodies to both cytokines inhibited the observed increase in ingestion. Together, these data demonstrate that ingestion of acapsular *C. neoformans* is mediated via mannose and β -glucan receptors on the macrophage

surface and that this process activates macrophages for enhanced phagocytosis of the encapsulated form via production of macrophage-derived cytokines.

AN 1995:659132 HCAPLUS <<LOGINID::20081212>>

DN 123:81423

OREF 123:14539a,14542a

TI Ingestion of acapsular *Cryptococcus neoformans* occurs via mannose and .
beta.-glucan receptors, resulting in cytokine production
and increased phagocytosis of the encapsulated form

AU Cross, C. E.; Bancroft, G. J.

CS Dep. Clinical Sciences, London Sch. Hygiene Tropical Med., London, WC1E
7HT, UK

SO Infection and Immunity (1995), 63(7), 2604-11

CODEN: INFIBR; ISSN: 0019-9567

PB American Society for Microbiology

DT Journal

LA English